UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/930,494	08/16/2001	Reid W. Von Borstel	1331-352	1560

23117 7590 11/19/2009

NIXON & VANDERHYE, PC
901 NORTH GLEBE ROAD, 11TH FLOOR
ARLINGTON, VA 22203

EXAMINER	
LEWIS, PATRICK T	

ART UNIT	PAPER NUMBER
1623	

MAIL DATE	DELIVERY MODE
11/19/2009	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte REID W. VON BORSTEL and JOEL A. SAYDOFF

Appeal 2009-006985
Application 09/930,494
Technology Center 1600

Decided: November 18, 2009

Before DONALD E. ADAMS, DEMETRA J. MILLS, and ERIC GRIMES, *Administrative Patent Judges*.

GRIMES, *Administrative Patent Judge*.

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 involving claims to methods of treating symptoms resulting from mitochondrial respiratory chain dysfunction. The Examiner has rejected the claims as obvious and nonenabled. We have jurisdiction under 35 U.S.C. § 6(b). We affirm-in-part.

STATEMENT OF THE CASE

The Specification discloses that "[m]itochondrial dysfunction contributes to various disease states" (Spec. 1). "In addition to congenital disorders involving inherited defective mitochondria, acquired mitochondrial dysfunction contributes to diseases, particularly neurodegenerative disorders associated with aging like Parkinson's, Alzheimer's, Huntington's Diseases" (*id.* at 2).

Claims 1-15, 18-41, and 47-49 are on appeal. Claims 1 and 37 are representative and read as follows:

Claim 1: A method for treating or preventing pathophysiological consequences of mitochondrial respiratory chain dysfunction in a mammal comprising administering to said mammal in need of such treatment or prevention an effective amount of a pyrimidine nucleotide precursor.

Claim 37: A method for treating developmental delay in cognitive, motor, language, executive function, or social skills in a mammal comprising administration of an effective amount of a pyrimidine nucleotide.

The claims stand rejected as follows:[1]

- Claims 1-15, 18-36, and 47-49 under 35 U.S.C. § 112, first paragraph, for lack of enablement; and

---

[1] The Examiner also provisionally rejected claims 1-15, 21, 23, 27, 31, 32, 37-41, and 47 for obviousness-type double patenting based on claims 48-59 of application 09/763,955 (Office Action mailed Apr. 12, 2006, page 3). Appellants have stated that they will file a terminal disclaimer to overcome this rejection (Appeal Br. 8). Since Appellants have not disputed the merits of the provisional obviousness-type double patenting rejection, we summarily affirm it.

• Claims 1-15, 18-32, and 37-41 under 35 U.S.C. § 103(a) as being obvious in view of Page[2] and von Borstel.[3]

ENABLEMENT

Issue

The Examiner has rejected claims 1-15, 18-36, and 47-49 under 35 U.S.C. § 112, first paragraph. With regard to claims 1-15, 18-32 and 47-49, the Examiner concludes that "the specification, while being enabling for the *treatment* of … pathophysiological consequences of mitochondrial respiratory chain dysfunction, does not reasonably provide enablement for the *prevention* of … pathophysiological consequences of mitochondrial respiratory chain dysfunction" (Ans. 3, emphases added). Similarly, with regard to claims 33-36, the Examiner concludes that "examples drawn to treatment [are] not sufficient to support the alleged applicability for the prevention of death or functional decline of postmitotic cells" (*id.* at 7).

Appellants contend that "the correct connotation of 'preventing' as used in the presently claimed invention … is the prophylactic administration of compounds of the invention which prevents progression or full manifestation of diseases related to essentially irreversible mitochondrial defects" (Appeal Br. 10). Appellants contend that the "data included in the present application involves both 'treatment' and 'neuroprotective' effects of a pyrimidine nucleotide precursor(s), used as a therapeutic for disorders involving mitochondrial respiratory chain enzyme impairment" (*id.* at 11),

---

[2] Page et al., *Developmental disorder associated with increased cellular nucleotidase activity*, 94 PROC. NATL. ACAD. SCI. USA, 11601-11606 (1997).
[3] Von Borstel et al., US 6,316,426 B1, Nov. 13, 2001.

and therefore the Specification is enabling for "prevention," as understood in the art (*id.* at 12).

The issue with respect to this rejection is: Has the Examiner shown that a person of ordinary skill in the art would not have accepted the Specification's disclosure to support enablement of preventing pathophysiological consequences of mitochondrial respiratory chain dysfunction or preventing death or functional decline of post-mitotic cells due to mitochondrial respiratory chain dysfunction?

Findings of Fact

1. The Examiner finds that the "treatment of diseases involving mitochondrial dysfunction is well known in the art. Treatment generally involves the administration of vitamins and cofactors used by particular elements of the mitochondrial respiratory chain." (Ans. 5.)

2. The Specification discloses "a method of preventing pathophysiological consequences of mitochondrial respiratory chain deficiency comprising administering to a mammal an amount of a pyrimidine nucleotide precursor effective in preventing the pathophysiological consequences" (Spec. 5).

3. The Specification discloses "methods for preventing or reducing death and dysfunction of postmitotic cells bearing mitochondrial respiratory chain deficits" (*id.*).

4. The Specification discloses that triacetyluridine attenuated taxol-induced neuropathy, which is associated with mitochondrial dysfunction, when administered to mice (*id.* at 45-46 (Example 5)).

5. The Specification discloses that triacetyluridine protects mice against central nervous system cell loss, weight loss, and mortality due to the infusion of azide, an inhibitor of cytochrome oxidase (*id.* at 55-57 (Example 12)).

Principles of Law

> When rejecting a claim under the enablement requirement of section 112, the PTO bears an initial burden of setting forth a reasonable explanation as to why it believes that the scope of protection provided by that claim is not adequately enabled by the description of the invention provided in the specification of the application; this includes, of course, providing sufficient reasons for doubting any assertions in the specification as to the scope of enablement.

In re Wright, 999 F.2d 1557, 1561-62 (Fed. Cir. 1993).

"Section 112 does not require that a specification convince persons skilled in the art that the assertions therein are correct." *In re Armbruster*, 512 F.2d 676, 678 (CCPA 1975).

"[D]uring examination proceedings, claims are given their broadest reasonable interpretation consistent with the specification." *In re Hyatt,* 211 F.3d 1367, 1372 (Fed. Cir. 2000).

Analysis

Claim 1 is directed to a method for treating or preventing pathophysiological consequences of mitochondrial respiratory chain dysfunction by administering a pyrimidine nucleotide precursor. Similarly, claim 33 is directed to a method for preventing death or functional decline of post-mitotic cells due to mitochondrial respiratory chain dysfunction by administering a pyrimidine nucleotide precursor.

The Examiner acknowledges that the Specification is enabling with respect to treating the conditions encompassed by claim 1, but finds that examples showing treatment are not "sufficient to support the alleged applicability for the prevention of … pathophysiological consequences of mitochondrial respiratory chain dysfunction" (Ans. 5). The Examiner also finds that examples showing treatment are not "sufficient to support the alleged applicability for the prevention of death or functional decline of postmitotic cells" (*id.* at 7).

Appellants argue that "[p]revention in the context of the present invention … applies to reducing the rate of progression of a chronic, worsening disease process compared with patients who do not receive the drug" and that "[i]n most cases, this will fall under the heading of 'treatment' of a diagnosed disease but, in other situations, e.g. where a genetic disorder has not yet (but eventually will) cause clinical symptoms …, the concept of prevention is medically and scientifically legitimate" (Appeal Br. 10-11).

We agree with Appellants that the Examiner has not adequately shown that the Specification does not enable preventing symptoms of mitochondrial respiratory chain dysfunction. Claim 1, given its broadest reasonable interpretation, does not require the prevention of all of the pathophysiological consequences, or the prevention forever, but only the prevention of some symptoms for some period of time. Likewise, the broadest reasonable interpretation of claim 33 requires only preventing the death or functional decline of *some* cells for *some* period of time.

The Examiner has acknowledged that the Specification is enabling for the *treatment* of pathophysiological consequences of mitochondrial respiratory chain dysfunction. The Specification states that the disclosed method is effective for both treatment and prevention of symptoms of mitochondrial respiratory chain dysfunction, and provides working examples that are said to show the prevention of at least some symptoms. The Examiner has provided no reasoning based on evidence or sound scientific reasoning to doubt the Specification's assertion. Thus, the Examiner has not adequately explained why one of skill in the art would conclude that the Specification is not enabling for the prevention of at least some of the symptoms of mitochondrial respiratory chain dysfunction for at least some period of time.

Conclusion of Law

The Examiner has not shown that a person of ordinary skill in the art would not have accepted the Specification's disclosure to support enablement of preventing pathophysiological consequences of mitochondrial respiratory chain dysfunction or preventing death or functional decline of post-mitotic cells due to mitochondrial respiratory chain dysfunction.

OBVIOUSNESS

Issue

The Examiner has rejected claims 1-15, 18-32, and 37-41 under 35 U.S.C. § 103(a) as being obvious in view of Page and von Borstel. Claims 1-15, 18-32, and 38-41 have not been argued separately and therefore stand or fall with claim 37. 37 C.F.R. § 41.37(c)(1)(vii).

The Examiner finds that Page discloses uridine for the treatment of patients having "developmental delay, seizures, ataxia, recurrent infections, severe language deficit, and an unusual behavioral phenotype characterized by hyperactivity, short attention span, and poor social interaction," but does not teach administration of an acyl derivative of uridine (as recited in claim 12) (Ans. 8).

The Examiner finds that "von Borstel teaches a family of uridine and cytidine derivatives," including "acyl derivatives for treating a variety of physiological and pathological conditions" (*id.* at 9). The Examiner concludes that it "would have been obvious to one of ordinary skill in the art … to treat patients having a mitochondrial disease with an acylated derivative of uridine" (*id.*).

Appellants contend that the Examiner erred because the cited references do not disclose or suggest "mitochondrial respiratory chain dysfunction as a molecular basis for symptoms" of the disease described in Page (Appeal Br. 15).

The issue with respect to this rejection is: Does the evidence of record support the Examiner's conclusion that the cited references disclose or suggest a method for treating developmental delay in, e.g., language or social skills by administering an effective amount of a pyrimidine nucleotide?

Additional Findings of Fact

6. Page discloses treatment of "four unrelated patients in whom developmental delay, seizures, ataxia, recurrent infections, speech deficit,

and an unusual behavioral phenotype were associated with highly elevated activity of cytosolic 5' nucleotidase" (Page 11601, right col.).

7. Page discloses that "[a]ll patients were markedly delayed in their developmental milestones, especially language" (*id.*).

8. Page discloses that "[a]ll four displayed an unusual behavioral phenotype that was characterized by . . . abnormal social interaction" (*id.*).

9. 109Page discloses that "[m]etabolic therapy with pyrimidine compounds appeared to be highly effective in reversing these manifestations" (*id.*).

10. Page discloses that patient 1 showed improvement with treatment with uridine monophosphate (UMP) and cytidine monophosphate (CMP), including improvement in speech: "Speech improved from short telegraphic sentences to longer, more complex, and age-appropriate expressions" (*id.* at 11604, right col.).

11. Page discloses that after treatment with UMP and CMP, patient 1's "[i]nteraction with others became more normal and appropriate for her age" (*id.*).

Principles of Law

"If the claim extends to what is obvious, it is invalid under § 103." *KSR Int'l Co. v. Teleflex Inc.*, 550 U.S. 398, 419 (2007).

"It is well settled that 'anticipation is the epitome of obviousness.'" *In re McDaniel*, 293 F.3d 1379, 1385 (Fed. Cir. 2002) (quoting *Connell v. Sears, Roebuck & Co.*, 722 F.2d 1542, 1548 (Fed. Cir. 1983)).

Analysis

Claim 37 is directed to a method for treating developmental delay in, for example, language or social skills in a mammal by administering an effective amount of a pyrimidine nucleotide. Page discloses treating a patient having developmental delay in language and social skills by administering an effective amount of UMP and CMP, pyrimidine nucleotides. Page therefore discloses the method of claim 37. The disclosure of von Borstel is not required to establish the unpatentability of claim 37; anticipation is the epitome of obviousness.

Appellants argue that the cited references do not disclose or suggest "mitochondrial respiratory chain dysfunction as a molecular basis for symptoms" of the disease described in Page (Appeal Br. 15).

This argument is not persuasive. Claim 37 is not limited to treating developmental delay that is caused by mitochondrial chain dysfunction; it reads on treating developmental delay in the recited areas regardless of the underlying cause.

Conclusion of Law

The evidence of record supports the Examiner's conclusion that the cited references disclose a method for treating developmental delay in language or social skills by administering an effective amount of a pyrimidine nucleotide.

SUMMARY

We affirm the provisional rejection of claims 1-15, 21, 23, 31, 32, 37-41 and 47 for obviousness-type double patenting and the rejection of claims

1-15, 18-32, and 37-41 under 35 U.S.C. § 103(a). However, we reverse the rejection of claims 1-15, 18-36 and 47-49 under 35 U.S.C. § 112, first paragraph, for lack of enablement.

TIME PERIOD FOR RESPONSE

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a).

AFFIRMED-IN-PART

lp

NIXON & VANDERHYE, PC
901 NORTH GLEBE ROAD, 11TH FLOOR
ARLINGTON VA 22203